No information on natural vectors is available for RWMV. CPsV is commonly transmitted by vegetative propagation and no natural vectors have been identified; in some cases natural spread of psorosis in limited citrus areas has been reported, but the spatial patterns would suggest a hypothetic aerial vector instead of a soil-borne one. The data are however based on symptom observation, not on analysis for the spread of CPsV.

For disease control the use of resistant or tolerant crops may be the best choice. In Japan, the use of resistant tulip cultivars is the most important component of managing TMMMV disease, as it can be highly effective and has no deleterious effect on the environment; resistance assays have allowed researchers to identify highly resistant tulip lines and use them for breeding new resistant cultivars. For lettuce in soil-less cultivation, using ultraviolet (UV) sterilization of nutrients has shown good results in prevention of MiLV and LRNV infection, although for field lettuces the prospect is less good as no classical sources of resistance or tolerance have yet been identified. In the case of CPsV, control of the sanitary status of mother plants for producing propagating material is essential. Shoot-tip grafting *in vitro* associated with thermotherapy or somatic embryogenesis from stigma and style cultures have been successfully used to eliminate CPsV from plant propagating material. Several transgenic citrus lines exist carrying parts of the CPsV genome, and promising resistance may emerge from these.

See also: Plant Rhabdoviruses.

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Papaya Ringspot Virus

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Introduction

The term papaya ringspot virus (PRSV) was first used in the 1940s to describe a viral disease of papaya. The name was used primarily to describe the ringspots that appeared on fruits from infected plants. Early investigations showed that the virus was transmitted by several species of aphids in a nonpersistent manner. That is, the aphid vector could acquire the virus in a short period of time while feeding on infected plants and likewise transmit the virus in a span of few seconds to less than a minute during subsequent feeding. In the same decade researchers from India and other places like Puerto Rico reported the occurrence of an aphid-transmitted disease of papaya; based on the symptoms on the leaves, it was identified as papaya mosaic virus. Work in the 1980s showed that the aphid-transmitted papaya mosaic virus and PRSV were really the same, and the name of PRSV was adopted. PRSV is a member of the family *Potyviridae*, a large and arguably the most economically important group of plant viruses. Today, the term papaya mosaic virus is reserved for a virus that is not aphid transmitted, belongs to the family *Potexviridae*, and causes the papaya mosaic disease which is seldom observed and not important commercially.

The systemic host range of PRSV is confined to plants in the families Caricaceae and Cucurbitaceae, with the primary economically important host being papaya and a range of cucurbits such as squash, watermelon, and melons.

It does cause local lesions on plants of the family Chenopodiaceae such as *Chenopodium quinoa* and *C. amaranticolor*. The disease on cucurbits was, early on, referred to as being caused by watermelon mosaic virus-1 (WMV-1). Later serological and molecular characterization showed that PRSV and WMV-1 are virtually identical. Based on their close relationship, a single name was adopted to unify both viruses into one group. The name PRSV was chosen due to its being named before WMV-1. To clarify host range, 'P' (PRSV-P) or 'P type' is used to designate virus infecting papaya and cucurbits, while 'W' (PRSV-W) or 'W type' refers to virus infecting cucurbits only. The virus symptoms on cucurbits are identical to those on Caricaceae. Leaves of infected plants show severe mosaic, and chlorosis, are deformed, and often exhibit shoestring-type symptoms. The fruits are also often deformed and bumpy. In papaya, PRSV infection is characterized by mosaic and chlorosis symptoms on leaves, water-soaked streaks on the petiole, and deformation of leaves that can result in shoestring-like symptoms that resemble mite damage (**Figure 1**). The virus can cause deformation and ringspot symptoms on the fruit, hence the name PRSV. Commercial PRSV-resistant transgenic papaya expressing the coat protein (*CP*) gene of the virus has been used to control PRSV P in Hawaii, as will be discussed later.

Figure 1 Symptoms of PRSV on papaya.

General Properties of PRSV

The virus particles are flexuous rods about 760–800 nm × 12 nm with single RNA of about 10 326 b in length. Virus particles consist of 94.5% protein and 5.5% nucleic acid by weight. It has a single coat protein (CP) of about 36 kDa. Analysis of purified virus preparations that are stored show that the CP degrades to smaller proteins of *c.* 31–34 and 26–27 kDa proteins, possibly due to proteolytic degradation. The density of the virion in purified preparations is 1.32 g cm^{-3} in CsCl.

PRSV should not be confused with another potyvirus, papaya leaf distortion mosaic virus (PLDMV), which occurs in Okinawa and other parts of Asia, such as Taiwan. This virus causes very similar symptoms as PRSV on papaya and cucurbits but is serologically unrelated and its CP shares only 55–59% similarity to that of PRSV.

PRSV Genome

A genetic map of PRSV genome with polyprotein processing sites and products is presented in **Figure 2** and their possible functions in **Table 1**. Much of the knowledge on the genome of PRSV has been obtained from extensive work done by the laboratory of Dr. Shyi-Dong Yeh of National Chung-Hsing University in Taiwan. The genomic RNA of PRSV is 10 326 nt in length excluding the poly(A) tract and contains one large open reading frame that encodes a polyprotein of 3344 amino acids starting at nucleotide position 86 and ending at position 10 120. A VPg protein is linked to the 5′ end of the RNA while a poly(A) tract is at the 3′ end. The polyprotein is cleaved into proteins designated (name (size in M_r)): P1 (63K), helper component (HC-Pro, 52K), P3 (46K), cylindrical

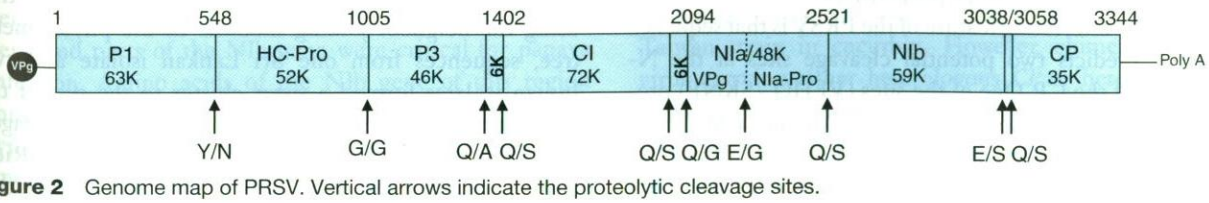

Figure 2 Genome map of PRSV. Vertical arrows indicate the proteolytic cleavage sites.

Table 1 PRSV proteins and their possible functions

Viral protein	*Size (M_r)*	*Functions*
P1	63K	Proteinase Cell-to-cell movement
HC-Pro	52K	Vector transmission Proteinase Pathogenicity Suppressor of RNA silencing Cell-to-cell movement
P3	46K	Unknown, but possible role in replication
6K1	6K	Unknown, but possible role in replication
CI	72K	Genome replication (RNA helicase) Membrane attachment Nucleic acid-stimulated ATPase activity Cell-to-cell movement
6K2	6K	Unknown, but possible roles in: • Replication • Regulation; inhibition of NIa nuclear translocation
NIaVPg	21K	Genome replication (primer for initiation of RNA synthesis)
NIaPro	27K	Major proteinase
NIb	59K	Genome replication (RNA-dependent RNA polymerase, RdRp)
CP	35K	RNA encapsidation Vector transmission Pathogenicity Cell-to-cell movement

inclusion protein (CI, 72K), nuclear inclusion protein a (NIa, 48K), nuclear inclusion protein b (NIb, 59K), coat protein (CP, 35K), as well as two other proteins 6K1 (6K) and 6K2 (6K). The cleaved proteins are arranged on the genome starting from the 5′ in order as: P1–(HC-Pro)–P3–6K1–CI–6K2–NIa–NIb–CP (**Figure 2**).

The cleavage proteins mentioned above have been identified by immunoprecipitation and dynamic precursor studies with PRSV-p as well as by extensive studies on proteolytic processing of polyproteins from other potyviruses. Three virus-encoded proteinases are responsible for at least seven cleavages: the P1 protein from N-terminus of the polyprotein autocatalytically liberates its own C-terminus, the HC-Pro also cleaves its own C-terminus, and NIa is responsible for *cis*- and *trans*-proteolytic processing to generate the CI, 6K, NIa, NIb, and CP proteins. NIa has also been shown to contain an internal cleavage site for delimitation of the genome-linked protein (VPg) and the proteinase (Pro) domains. Thus, the genomic organization and processing of the polyprotein of PRSV is similar to those of other potyviruses.

A rather interesting feature of the PRSV is that sequence analysis predicts two potential cleavage sites at the N-terminus of the CP. One of the sites (VFHQ/SKNF) predicts a CP of 33K and a NIb of 537 amino acids about 20 amino acids larger than those of other potyviruses. The second predicted cleavage site (VYHE/SRGTD) generates a CP of 35K and an NIa of 517 amino acids. There is no firm evidence to suggest that only one cleavage site is used. If both sites are used in polyprotein processing, one would expect heterogeneous products. This may explain why the analysis of purified CP preparations that are stored frequently shows the major ~36K form in addition to smaller CPs that are 2–5K smaller.

Sequence Diversity and Evolution

Knowledge of the sequence diversity among isolates of a virus has great implications in developing an effective virus disease management program and in understanding the origin and biology of the virus. Recently, numerous PRSV P and W sequences from virus isolated from different parts of the world have been reported in the sequence database. Amino acid and nucleotide sequence divergence among PRSV isolates differ by as much as 14%. These differences, interestingly, are considerably less than that found among isolates of other potyviruses, such as yam mosaic virus (YMV), that differ by as much as 28%. Although initial data from the USA and Australia suggested that there was little variation among PRSV isolates within these countries, more recent data from India and Mexico have suggested that the sequence variation between PRSV isolates in other countries may be greater than previously recognized. Heterogeneity in *CP* length ranging from 840 to 870 nt has been noted. The observed size differences in *CP* sequence occurred in multiples of three, preserving the reading frame between genes of different genomes and resulting in CPs of between 280 amino acids (Indian P isolate KA2) and 290 amino acids (VNW-38 from central Vietnam). Interestingly, the CP-coding region of all isolates from Thailand were 286 amino acids in length, while those from India and Vietnam demonstrated considerable heterogeneity in CP length, at 280–286 and 285–290 amino acids, respectively. The first 50 or so amino acids of the N-terminal region of the PRSV *CP* gene were found to be highly variable and all differences in CP length were confined to this region. The differences in this region that did exist consisted of conservative amino acid substitutions. The majority of the size differences occurred in one of two hypervariable regions and most were due to differences in the number of EK repeats.

A phylogenetic study based on *CP* sequences from 93 isolates of type P and W PRSV from different geographic locations was done by generating a phylogenetic tree using the neighbor-joining method. In the phylogenetic tree, sequences from one Sri Lankan isolate and two Indian isolates formed a sister cluster to the rest of the sequences. The other isolates formed two major lineages: I included all isolates from the Americas, Puerto Rico, Australia, and a few from South Asia; and II included

isolates from Southeast Asia and the Western Pacific. Lineage I was the major of the two, containing three clusters of Brazilian isolates, two Indian isolates, and Australian, Mexican and US isolates. Within lineage I, the Brazilian and Mexican isolates were more diverse than the US and Australian isolates. Lineage II included all of the isolates from the Southeast Asia and Western Pacific, including China, Indonesia, Thailand, Vietnam, Taiwan, Japan, and the Philippines. However, the subclustering of isolates did not correlate well with their geographic origins; rather, they appeared to be a single mixed population with some well-defined subpopulations. These observations suggest that considerable movement of PRSV isolates has occurred among the Southeast Asian countries. Thai isolates of the P type diverged together, whereas PRSV W type diverged with other Southeast Asian isolates. Both P and W isolates of PRSV from Vietnam were intermingled with other Asian isolates. Sequence analysis showed that all Vietnamese isolates (except the P type from the southern part of the country) diverged from a common branch with P isolates from Japan and Taiwan while PRSV isolates from South Vietnam were diverged compared to those from the Philippines and seemed closely related to several W types from Thailand.

An interesting feature of PRSV is the origin of types P and W. As noted above, a number of viral diseases on cucurbits were historically associated with WMV-1 and not with any diseases of papaya. Did type P originate from W, or was it the reverse, or did they evolve independently? Evidences from various sources indicate that PRSV is primarily a pathogen of cucurbits, and that PRSV P originated from PRSV W. Work in Australia suggests that the recent outbreak of PRSV P came from the population of PRSV W already present in Australia. This suggestion is also supported by the diversity in cucurbit-infecting potyviruses that are phylogenetically related to PRSV.

Infectious Transcripts of Recombinant PRSV Help to Reveal Potential Determinants of Several Biological Characteristics

Determinants for Host Range Specificity

Studies utilizing the technique of producing infectious transcripts from recombinant viruses followed by bioassays of the transcripts have demonstrated that sequences of the PRSV genome responsible for determining papaya and cucurbit host specificity are not in the region of the *CP* gene. However, nucleotides 6509–7700 encoding the NIa gene and parts of the NIb gene were critical for papaya infection. Amino acids of the NIb gene of this region (nucleotides 7644–7700) between PRSV-P and PRSV-W type are identical, whereas sequence comparison of nucleotides 6509–7643 of four type P and two type W viruses showed that two amino acids at positions 2309 (K → D) and 2487 (I → V) of PRSV are significantly different between papaya-infecting type P and non-papaya-infecting type W. Further point mutational studies in these sites indicated that these two amino acids located in the NIa proteinase are responsible for conferring the ability to infect papaya.

Determinants for Local Lesion Formation on *Chenopodium*

As noted earlier, PRSV causes local lesions on *C. amaranticolor* and *C. quinoa*. The severe strain PRSV HA strain from Hawaii causes local lesions on *C. quinoa* but a mild nitrous acid mutant of it, PRSV HA 5-1, does not. Recombinant infectious viruses were generated by exchanging genome parts between PRSV HA and PRSV HA 5-1. The study revealed that the pathogenicity-related region is present between nucleotide positions 950 and 3261 of the PRSV HA genome and mutations in the *P1* and *HC-Pro* genes resulted in the attenuation of PRSV HA symptoms and the loss of ability to produce local lesions on *C. quinoa*. The *HC-Pro* gene of PRSV is the major determinant factor for local lesion formation.

Determinants on Severity of Symptoms, Suppression of Gene Silencing, Infection of Transgenic Papaya

Virus–host interaction studies based on recombinant analyses between severe and mild strains of PRSV indicated that the *HC-Pro* gene plays an important role in viral pathogenicity and virulence and acts as a suppressor of the gene-silencing defense mechanism in the papaya host plant. In addition, the comparative reaction of recombinant PRSV with chimeric *CP* gene sequences showed that heterologous sequences and their position in the *CP* gene influences their pathogenicity on PRSV-resistant transgenic papaya.

An interesting phenomenon of strain-specific cross-protection was observed in papaya and horn melon provided by the mild strain HA 5-1 of PRSV. The PRSV mild mutant HA 5-1 provided 90–100% protection against the severe parental strain PRSV HA in greenhouse and field conditions. However, the degree of protection provided in horn melon by HA 5-1 against a PRSV type-W strain from Taiwan was only 20–30%. Studies on strain-specific cross-protection phenomenon indicated that the recombinant HA 5-1 carrying both the heterologous *CP* and the 3′ untranslated region (UTR) of the PRSV W from Taiwan significantly enhanced the protection against the Taiwan strain in cucurbits. However, chimeric HA 5-1 virus carrying either heterologous *CP* or heterologous 3′ UTR showed reduced effectiveness of protection against PRSV HA in papaya when compared to protection by the native mild HA 5-1.

Similar to other potyviruses, PRSV is also transmitted by insect vector aphids (*Myzus persicae* and *Aphis gossypii*) in a nonpersistent manner. Detailed studies with other potyviruses show that HC-Pro/virions interaction is essential for aphid transmission of potyviruses. Although not empirically tested, it would seem likely that aphid transmission of PRSV would similarly be governed by HC-Pro/virion interactions.

Pathogen-Derived Resistance for Controlling PRSV: The Hawaii Case

The control or management of PRSV has been approached through practices such as quarantine, eradication, avoidance by planting papaya in areas isolated from the virus, continual rogueing of infected plants, use of tolerant lines to reduce damage caused by PRSV, cross-protection through the use of mild virus strains, and resistance using the approach of 'pathogen-derived resistance'. The efforts to control PRSV in Hawaii are described because it involves all of the above practices. Ultimately, the most successful has been the 'pathogen-derived resistance' approach.

The state of Hawaii consists of eight main islands that are in rather close proximity to each other with the shortest and farthest distance between islands being 7 mile between Maui and Kahoolawe and 70 mile between Kauai and Oahu. Travels between the islands are prevalent with the exception of Kahoolawe, which is not inhabited, and Niihau, which is privately owned. PRSV was first detected in the 1940s on Oahu where Hawaii's papaya industry was located at that time. Efforts to control the virus on Oahu largely consisted of state officials and farmers continually monitoring for infected plants and rogueing them, especially in areas where the virus was not prevalent. However, by the late 1950s, PRSV was causing extensive damage, which caused the papaya industry to relocate to Puna on the island of Hawaii.

The relocation of the industry to Puna was timely and effective because Puna had an abundance of land that was suitable to grow Kapoho, a cultivar of excellent quality that adapted to the volcanic soil base there, allowing excellent drainage, had high rainfall and yet lots of sunshine, and the land there could be bought or leased at reasonable prices. By the 1970s, the Kapoho papaya grown in Puna accounted for 95% of the state's papaya production, making papaya the second most important fruit crop behind pineapple.

Despite strict quarantine on movement of papaya seedlings between islands, PRSV was discovered in the town of Hilo which was only about 18 miles away from the center of the papaya-growing area of Puna. However, PRSV was indeed discovered in Puna in May 1992 (**Figure 3(a)**) and the Hawaiian papaya industry would be forever changed. By 1995, a third of the papaya growing area was completely infected and much of the rest of Puna had widespread infection (**Figure 3(b)**). By 1998, the production of papaya in Puna had dropped to 27 million pounds of papaya from 52 million pounds in 1992 when PRSV was discovered in Puna. In retrospect, the efforts of quarantine, monitoring and rogueing of infected plants in Hilo, and suppression efforts of PRSV in Puna all played key roles in helping Hawaii's papaya industry, because it gave researchers time to develop control measures for PRSV.

Research to develop tolerant varieties and cross-protection measures were started in the 1970s. Since resistance to PRSV has not been identified in *Carica papaya*, researchers have used tolerant germplasm in an attempt to develop papaya cultivars with acceptable PRSV tolerance and horticultural characteristics. However, tolerance to PRSV is apparently governed by a family of genes that is inherited quantitatively, which makes it technically difficult to develop cultivars of acceptable horticultural quality. Furthermore, the tolerant lines do become infected with PRSV, although fruit production continues still at a lower level. Indeed, in Thailand, the Philippines, and Taiwan, a number of tolerant lines have been developed and are used. However, Hawaii grows the small 'solo'-type papaya and efforts to introduce tolerance into acceptable 'solo' papaya cultivars have not been successful.

Efforts to use cross-protection were similarly started in the late 1970s to control PRSV in Hawaii. Cross-protection can be defined as the use of a mild strain of virus to infect plants that are subsequently protected against economic damage caused by a severe strain of the same virus. This practice has been used successfully for many years to minimize damage by citrus tristeza virus in Brazil, for example. In the early 1980s, a mild strain of PRSV (described above as PRSV HA 5-1) was developed through nitrous acid treatment of a severe strain, PRSV HA isolated from Oahu island. This mild strain was tested in Hawaii on Oahu island and showed good protection against damage by severe strains but produced symptoms that were very obvious on certain cultivars, such as Sunrise, especially in the winter months. This prominent symptom induction on certain cultivars and the logistics of mild strain buildup and inoculation of plants, among others, were factors that caused it not to be consistently used on the island of Oahu. There was no justification to use it on the island of Hawaii because PRSV was not yet found in Puna during the 1980s. Interestingly, the mild strain was used extensively for several years in Taiwan, but it did not afford sufficient protection against the severe strains from Taiwan and thus its use was abandoned after several years.

Transgenic Resistance

In the mid-1980s, an exciting development on tobacco mosaic virus (TMV) provided a rationale that resistance

Figure 3 (a) Healthy Puna papaya in 1992. (b) Severely infected papaya orchards in Puna in 1994. (c) Field trial of transgenic papaya. PRSV-infected nontransgenic papaya on left and PRSV-resistant transgenic papaya on right. (d) Commercial planting of transgenic papaya one year after releasing seeds of PRSV-resistant transgenic papaya. (e) Transgenic papaya commonly sold in supermarkets. (f) Risk of growing nontransgenic papaya still exists in 2005. Foreground is PRSV-infected nontransgenic papaya that are cut, and background shows healthy PRSV-resistant transgenic papaya.

to plant viruses could be developed by expressing the viral *CP* gene in a transgenic plant. This approach was called CP-mediated protection, and, at about the same time, a report introduced the concept of 'parasite-derived resistance'. The report on transgenic resistance to TMV set off a flurry of work in many laboratories to determine if this approach could be used for developing resistance to other plant viruses. Likewise, work was initiated in 1985 to use this approach for developing PRSV-resistant transgenic papaya for Hawaii.

Key requirements for successful development and commercialization of transgenic virus-resistant plants are the isolation and engineering of the gene of interest, vectors for mobilization into and expression of the gene in the host, transformation and subsequent regeneration of the host cells into plants, effective and timely screening of transformants, testing of transformants, and the ability to deregulate and commercialize the product.

The *CP* gene of the mild strain of PRSV was chosen as the 'resistance' gene because it had been recently cloned and it was of the PRSV P type. The gene was engineered into a wide host range vector that could replicate in *Escherichia coli* as well as in *Agrobacterium tumefaciens*, the bacterium used for one of the most widely used methods of plant transformation. The commercial cultivars Kapoho, Sunrise, and Sunset were chosen for transformation. Initially, transformation of papaya was attempted using the *Agrobacterium*–leaf piece approach, where leaf pieces would be infected with *Agrobacterium* harboring the *CP* gene and transformed cells would be regenerated via organogenesis into transgenic plants. The latter is the direct regeneration of cells from an organ such as the leaf. This approach did not work due to our failure to develop plants from leaf pieces. A shift to the transformation of somatic embryos via the biolistic (often referred to as the gene gun) approach resulted in obtaining of about a dozen transgenic papaya lines, four of which expressed the *CP* gene. In 1991, tests of the R_0 lines identified a transgenic Sunset that expressed the *CP* gene of PRSV HA 5-1, and showed resistance to PRSV from Hawaii. A field trial of R_0 plants was started in April 1992 on as the island of Oahu, and a month later PRSV was discovered at Puna in May 1992, as discussed above.

The Oahu field trial showed that R_0 plants of line 55-1 were resistant, and line 55-1 was further developed to obtain the cultivar 'SunUp' which is line 55-1 that has the *CP* gene in a homozygous state, and 'Rainbow' which is an F_1 hybrid of SunUp and the nontransgenic 'Kapoho'. SunUp is red-fleshed and Rainbow is yellow-fleshed. In 1995, SunUp and Rainbow were tested in a subsequent field trial in Puna and showed excellent resistance (**Figure 3(c)**). Due to its yellow flesh and good shipping qualities, Rainbow was especially preferred by the growers. Line 55-1 was deregulated by the US government and commercialized in May 1998. The deregulation also applied to plants that were derived from line 55-1. The timely commercialization of the transgenic papaya in 1998 was crucial since PRSV had decreased papaya production in Puna by 50% that year compared to 1992 production levels. The transgenic Rainbow papaya was quickly adopted by growers and recovery of papaya production in Hawaii was underway (**Figure 3(d)**). The transgenic papaya is sold throughout Hawaii (**Figure 3(e)**) and the mainland USA, and to Canada where it was deregulated in 2003. However, several challenges remain: coexistence, exportation of nontransgenic papaya to Japan, deregulation of transgenic papaya in Japan, and the adoption of transgenic papaya in other countries that suffer from PRSV.

Hawaii still needs to grow nontransgenic papaya to satisfy the lucrative Japanese market as well as for production of organic papaya, for example. Interestingly, the islands of Kauai and Molokai do not have PRSV but grow only limited acreage of papaya. This situation illustrates the point that many factors influence decisions on the localities and crops that are grown. In Hawaii, Puna is the best place to grow papaya for the reasons mentioned above; there is a lot of land, farmers there are intuned to growing the crop, the region receives plenty of water, sunshine, and has a well-drained lava-based 'soil' structure, and there are high-quality cultivars adapted to the local growing conditions. The disadvantage is PRSV, but that disadvantage was overcome through the introduction of the PRSV-resistant Rainbow papaya that has good commercial attributes plus virus resistance. Puna accounts for 90% of Hawaii's papaya and, as of 2005, Rainbow represents 66% of the papaya grown in Puna. Growing nontransgenic papaya can be risky because PRSV is still around (**Figure 3(f)**), but judicious use of isolation from virus sources and constant rogueing can provide a means of raising nontransgenic papaya. However, a major market is Japan, which still has not deregulated the transgenic papaya. To maintain the lucrative Japanese market, Hawaii has to continue the exportation of nontransgenic papaya to Japan. The immediate solution is to concurrently grow nontransgenic and transgenic papaya, and subsequently to deregulate the transgenic papaya so it can be freely shipped into Japan. What approaches are being taken?

Currently, Japan accepts nontransgenic papaya but it needs to be free of 'contamination' by transgenic papaya. The Hawaii Department of Agriculture (HDOA) and the Japan Ministry of Agriculture, Forestry and Fisheries (MAFF) have agreed on an 'identity preservation protocol' (IPP), in which nontransgenic papaya can even be grown in close proximity (coexistence) to transgenic papaya in Puna, for example, and still be shipped to Japan. The protocol involves a series of monitoring and checkpoints in Hawaii that allow direct marketing of the papaya without delay while samples of the shipment are spot-checked by MAFF officials in Japan. The process has worked very well and allowed Hawaii to maintain its market share in Japan. This represents a practical case of 'coexistence' of transgenic and nontransgenic papaya and fruitful collaboration between governments (Hawaii and Japan) that provides mutual benefits to all parties.

The ideal situation would be, however, to freely export nontransgenic and transgenic papaya to Japan. To this end, efforts are underway to deregulate the transgenic papaya in Japan by obtaining approval from Japanese governmental agencies such as MAFF, the Ministry of Health, Labor, and Welfare (MHLW), and the Ministry of the Environment

(MOE). MAFF has provisionally approved Hawaii's transgenic Rainbow and SunUp papayas, and efforts to present the final documentation to all three agencies are nearing completion. Deregulation of the transgenic papaya in Japan not only would expand the Hawaiian transgenic market but would also be a good case study for evaluating the effectiveness of commercialization and marketing of fresh transgenic products, since the transgenic papaya in Japan would be labeled, and subsequently consumers there would be given an opportunity to make a personal choice between a transgenic and nontransgenic product. The implications of this opportunity are obvious given the current 'controversial' climate over genetically modified organisms (GMOs) in the world. It is indeed rare that the previously little known PRSV could perhaps provide an example that would help us to resolve such controversies.

Summary Remarks

PRSV has been thoroughly characterized and is a typical member of the family *Potyviridae*, arguably the largest and economically most important plant virus group. The complete genome sequence has been elucidated and infectious transcripts have provided a means to determine the genetic determinants of some important biological functions such as host range and virulence. Furthermore, pathogen-derived resistance has been used to control PRSV in Hawaii through the use of virus-resistant transgenic papaya. In the US, only three virus-resistant transgenic crops have been commercialized: squash, papaya, and potato. The transgenic virus-resistant papaya provides a potential means to test the global acceptance of GMOs while presenting a plausible approach to control a disease affecting papaya worldwide.

See also: Plum Pox Virus; Potato Virus Y; Plant Resistance to Viruses: Engineered Resistance; Watermelon Mosaic Virus and Zucchini Yellow Mosaic Virus.

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Pecluvirus

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Glossary

Coiled-coil motif A protein structure in which two to six α-helices of polypeptides are coiled together like the strands of a rope.
Hetero-encapsidation Partial or full coating of the genome of one virus with the coat protein of a differing virus. Also termed transcapsidation or heterologous encapsidation.
Leaky scanning mechanism Mechanism by which the ribosomes fail to initiate translation at the first AUG start codon, and scan downstream for the next AUG codon.
Post-transcriptional gene silencing Mechanism for sequence-specific RNA degradation in plants.
t-RNA-like structure Structure mimicking a t-RNA.
Virus-like particles Consist of the structural proteins of a virus. These particles resemble virions meaning that they are not infectious.

History

Pecluviruses, responsible for the 'clump' disease in peanut (=groundnut, *Arachis hypogaea*), have been reported from